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The UCLA Population Studies of Chronic Obstructive Respiratory Disease

VII. Relationship between Parental Smoking and Children's Lung Function[1-3]

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SUMMARY Results of previous studies of the impact of parents' smoking on children's lung function have been conflicting. To evaluate further the effect of passive smoking on the lung function of children, we analyzed respiratory questionnaire and lung function results obtained during field testing of residents (aggregated according to household) of 4 census tracts in the Los Angeles area. We report here on 971 white, non-Hispanic, nonsmoking, nonasthmatic children residing in households in which the smoking status of both parents was known; households with ex-smoking parents were excluded from analysis. We divided these children into 3 categories related to parental smoking status: (*1*) at least mother smokes, (*2*) only father smokes, and (*3*) neither parent smokes. Prediction equations for several indexes of forced expired volume and flow were derived separately for boys and girls 7 to 17 yr of age. Analysis of variance was used to compare lung function residuals of children in the 3 different passive smoking categories. Analysis was performed separately on younger (7 to 11 yr of age) and older (12 to 17 yr of age) children of each sex. Among younger male children, residual values were significantly lower in the maternal smoking category than in the other 2 household categories for maximal flow and maximal flow after exhalation of 25% of forced vital capacity (FVC) ($p < 0.05$); no differences were noted between the paternal-smoking only and nonsmoking household categories. A trend toward similar results was found in older male children, which approached significance ($p < 0.1$). Among females, forced expiratory flow during the middle half of the FVC and maximal flow after exhalation of 75% of FVC were significantly lower in relation to maternal smoking in the older children only ($p < 0.05$). ANOVA revealed no decrement in lung function in relation to passive smoking among children with asthma or bronchitis ($n = 138$). No differences were noted by chi-square analysis in the frequency of any respiratory symptom comparing children in the different passive exposure categories. We conclude that passive exposure to at least maternal smoking (but not to paternal smoking alone) affects the airways of younger boys. The apparent effect on the lung function of heavily exposed older girls is more likely to be confounded by selective underreporting of "active smoking."

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Introduction

Habitual inhalation of the combustion products of tobacco is known to adversely affect the health of active (voluntary) smokers (1). Considerably less is known about how tobacco smoke affects the health of involuntarily exposed nonsmokers. Regular tobacco smoking produces readily detectable alterations in the structure and function of the respiratory tract of active smokers. Sidestream smoke contains many of the same constituents found in the smoke inhaled by voluntary smokers. Thus, the frequent involuntary inhalation of air contaminated by tobacco smoke may produce qualitatively similar, but quantitatively fewer, alterations in the lungs of passive smokers. The extent of such changes would be expected to depend on a number of factors. These include (*1*) the intensity of exposure, which is a function of characteristics of the environment (e.g., room volume and ventilation), the number of cigarettes smoked per unit time, proximity to the source of cigarette smoke, and the ratio of sidestream to exhaled, mainstream smoke; and (*2*) the characteristics of the person exposed (e.g., inherent airways sensitivity to constituents of tobacco smoke, and age). Because younger children have immature lungs, less immunity to respiratory infections, and a greater minute ventilation relative to body weight, they may be more vulnerable to adverse pulmonary effects of passive smoking.

Previous studies have investigated possible effects of passive smoking on the occurrence of respiratory infections and symptoms in infants and children with variable results. A relationship has been found between parental smoking and the incidence of lower respiratory illness in children, but only or mainly during the first year of life (2-7). Children exposed to parental smoking have been found to have a greater frequency of respiratory symptoms than unexposed children (8-11). However, in some studies (8-10), this difference was no longer significant when adjustment was made for parents' respiratory symptoms, possibly reflecting transmission of infection to children or a bias towards reporting symptoms by symptomatic parents. In contrast, an apparent dose-dependent relationship was found between parental smoking and persistent wheeze in children, which was independent of the presence of wheezing in either parent (11).

Studies of the relationship between parental smoking and children's lung function have also yielded variable results. Schilling and coworkers (10) failed to find any significant relationship between parents' smoking and

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several indexes of lung function derived from timed spirometry in boys 7 to 17 and girls 7 to 14 yr of age, except for maximal flow after exhalation of 50% of forced vital capacity ($\dot{V}max_{50}$) in girls in households where only the mother smoked. On the other hand, Tager and associates (12) detected a clear trend toward an inverse relationship between the level of predicted forced expiratory flow during the middle half of the forced vital capacity (FEF_{25-75}) as a ranked score in children 5 to 9 yr of age and the number of smoking parents in the household, although this relationship was not statistically significant. The discrepancy in findings between these 2 groups of investigators may be explained by differences in the study populations, the actual levels of exposure to passive smoking, and/or the methods of analysis.

In view of the conflicting findings of previous workers concerning the possible impact of passive exposure to sidestream smoke on respiratory symptoms and lung function of nonsmoking children, we further evaluated this question by analyzing respiratory questionnaire and lung function data collected during field testing of residents (aggregated according to household) of 4 census tracts in the Los Angeles area.

Methods

A cohort study of chronic obstructive respiratory disease has been under way in 4 selected areas of Los Angeles County since 1973 (13–17). The areas were selected to be homogeneous in demographic characteristics, and heterogeneous across sites in historical exposure to different levels and types of atmospheric pollutants. All 4 areas are predominantly white and middle class. Residents in over 90% of households in each area were enumerated. All persons 7 yr of age and older were invited to be interviewed and undergo lung function testing in a mobile laboratory located in the selected areas. The interview schedule consisted of a modified National Heart, Lung and Blood Institute questionnaire (13, 14) and provided demographic data and information on symptomatology, respiratory and allergic disease history, smoking behavior (present and past cigarette use, pipe and cigar consumption), history of occupational exposure to hazardous substances, and residence history. Responses of children 12 yr of age or younger to questions concerning smoking behavior were not thought to be reliable under the conditions of this study in which younger children usually accompanied their parents to the mobile laboratory; therefore, these children were all assumed to be nonsmokers. Although similar reservations were held concerning the reliability of responses of teenage children to the same questions, the older children were asked to respond to these questions. Usually (but not always), the teenage children's parents were not present during the interview. Lung function measurements included tests of forced exhalation (timed spirometry, maximal expiratory flow-volume curves), single-breath nitrogen washout, and whole body plethysmography (airway resistance, thoracic gas volume at functional residual capacity, and specific airway conductance). Data reported in this study were collected between 1973 and 1977 during the first round of testing in each of the 4 study areas.

Households with children 7 to 17 yr of age were identified from the household rosters. Only households in which one or both parents were regular smokers or lifetime nonsmokers were included in the analysis. Households in which one or both parents were former smokers were excluded because of wide variability in the duration of abstinence from tobacco among ex-smoking parents and, hence, in the passive smoking exposure of their children. Households with nonparent adults who were regular or former smokers or whose smoking status was unknown were excluded from analysis, except if both parents in the household were already regular smokers (see below). Regular cigarette smokers were defined as persons who reported that they currently smoked more than 1 cigarette per day on a regular basis and had done so for at least 1 yr. Nonsmokers were defined as those who had *never* smoked more than 1 cigarette per day on a regular basis for at least 1 yr. Only white children *without* a history of asthma, bronchitis, or other reported lung disease who did not indicate that they had ever smoked were included in the main analyses. White children *with* asthma or bronchitis who were self-reported never-smokers comprised a separate group for analysis.

Our sample was derived in the following manner. Households were excluded either because they failed to meet the stated criteria or because necessary information was missing on one or more household members. An enumerator visited 9,083 households. In 5,533 of these, all the members of the household 7 yr of age or older participated in the study. Twenty-seven percent of these households, or 1,476, had children in the 7- to 17-yr age range who underwent pulmonary function testing in the mobile laboratory. After excluding households with former smokers, and households that contained at least 1 nonparent adult who smoked (or whose smoking status was unknown) in an otherwise nonsmoking household or in one where only one parent smoked, 840 households with 1,472 children were eligible for study. Children were excluded who were nonwhite or had a Spanish surname. In addition, children who were known ever-smokers or had physician-diagnosed asthma, bronchitis, or other reported lung disease, were excluded from the main analysis. This left 1,160 children for study. An additional 189 children were excluded because questionnaire or lung function data were incomplete and/or missing, or because their maximal expiratory flow-volume curves were technically unsatisfactory. A total of 971 children from 615 households were thus eligible for study. These children represent 28% of all children 7 to 17 yr of age in the study cohort and 66% of such children who resided in households that were eligible for study. An additional 138 white, reportedly never-smoking children *with* a physician-diagnosis of asthma or chronic bronchitis were analyzed separately.

Analyses reported here, therefore, are for 3 types of households: (*1*) at least mother smokes, (*2*) only father smokes, and (*3*) no one smokes. In all analyses, age, height, and area were controlled for in a linear fashion. Analyses were also performed separately for male and female children and for 2 age-defined subgroups of children: those 7 to 11 and those 12 to 17 yr of age. This latter subdivision was made for 2 reasons. First, it was desirable to see if younger or older children were more affected by parental smoking. Second, although no children were included in the analyses who were self-reported smokers, there was concern that some teenagers who actually smoked might not report their smoking to an interviewer. Families often came together to the mobile function laboratory and the members were processed consecutively. Knowing that their parents were nearby might have influenced some teenage smokers to say that they did not smoke. On the other hand, younger children (even though not questioned as to their smoking behavior) are much less likely to smoke, particularly in these middle-class areas (1).

Linear regression equations predicting each of 7 indexes of lung function were run separately for male and female children: FVC, forced expiratory volume in one second (FEV_1), FEF_{25-75}, $\dot{V}max$, $\dot{V}_{25}$, $\dot{V}_{50}$ and $\dot{V}_{75}$. Age, height, and geographic area (dummy variable) were entered as predictor variables. These equations were obtained for children without self-reported chronic lung disease who came from households where everyone was nonsmoking. Residuals were calculated for all male and female children, using the appropriate equation, regardless of the smoking status of their parents. One-way analysis of variance (18) was used to test whether residual lung function results by sex were equal in the 3 types of households. Mean residual levels are set at zero for children who came from nonsmoking households (who were used to define the equations). The size of the mean residual for the other 2 groups is an indicator of the effect parental smoking has on these children. One-way analyses of vari-

TABLE 1

NUMBER OF CHILDREN 7 TO 17 YR OF AGE BY HOUSEHOLD TYPE

Household Type	Male Children (n)	Female Children (n)	Overall (n)
At least mother smokes	207	200*	407†
Only father smokes	99	93*	192
Everyone nonsmoking	190*	182*	372
Total	496	475	971

* For FEF_{25-75}, $\dot{V}max$, $\dot{V}_{25}$, $\dot{V}_{50}$ and $\dot{V}_{75}$, males $n = 188$ (everyone nonsmoking), females $n = 198$ (at least mother smokes), $n = 92$ (only father smokes), and $n = 180$ (everyone nonsmoking). For definition of abbreviations, see table 2.

† Of the 407 children whose mother smokes, 276 come from households where both parents smoke, and 131 come from households where the father does not smoke.

ance also were performed on residuals across household types within the 2 age groups (7 to 11 and 12 to 17 yr of age). Mean residual levels for children from nonsmoking households within these separate groups will not necessarily be zero because prediction equations were based on data from both age groups combined. These same analyses were repeated, using one-way analysis of covariance, where age, height, and geographic area were entered as covariates.

In our analyses, which assume independent observations, we did not adjust for sibship size. Therefore, a portion of our observations lacks independence. However, because our data were analyzed separately by sex, this portion is not large, 23% for males and 20% for females. Moreover, when the data are further divided into age groups 7 to 11 and 12 to 17 yr of age, the portion of the households that contributes more than one child to each analysis is even less. Therefore, our analyses were for the most part based on independent observations.

For those lung function results in which the analysis of variance indicated a significant difference among the mean residual levels for the 3 types of households, linear contrasts were performed (19). Two contrasts were uniformly performed. First, the means for nonsmoking households were contrasted with those for households where only the father smoked. Second, the means for households where at least the mother smoked were contrasted with the pooled means of the other 2 household categories. The significance level, or p value, is presented using the t test. This p value is compared to an α of 0.025 for an overall level of significance of 5% for 2 contrasts, or 0.05 if only 1 contrast is considered (19). In accordance with standard reporting procedures, the significance levels we used apply only to 1 lung function test for each set separately, not across all lung function tests.

Prevalence of each type of respiratory symptom (cough, sputum, increased cough and sputum, wheezing, breathlessness, acute chest illness) or of *any* respiratory symptom among children from the 3 types of households was compared using chi-square or Fisher's exact test (20). Individual children were judged to have a positive history for each of the above symptoms according to previously published criteria (13, 14).

Results

A total of 971 children 7 to 17 yr of age were studied in the 4 geographical areas. The number of male and female children in each of the 3 types of households is given in table 1. Thirty-eight percent of the children resided in households in which no one reported smoking. If only one parent smoked, 59% of the time it was the father. Over 90% of the smoking parents started smoking before the birth of the oldest of their children included in our analysis. The average ages and heights of the children were very similar by type of household.

The mean values of adjusted lung function obtained from covariance analysis, lung function residuals, and the results of the analyses of variance computed on the residuals across household types within sex are reported in table 2. For male children 7 to 17 yr

TABLE 2

ADJUSTED MEAN VALUES OF CHILDREN'S LUNG FUNCTION,* RESIDUAL LUNG FUNCTION,† AND SIGNIFICANCE LEVELS,† BY HOUSEHOLD TYPE AND SEX OF CHILD

Lung Function Test, Unit	Male Children			Female Children			Overall Mean		Level of Significance†	
	At Least Mother Smokes	Only Father Smokes	Everyone Nonsmoker	At Least Mother Smokes	Only Father Smokes	Everyone Nonsmoker	Males	Females	Males	Females
Adjusted mean lung function*										
FVC, L	2.84	2.92	2.96	2.54	2.57	2.53	2.90	2.54		
FEV_1, L	2.41	2.51	2.50	2.20	2.26	2.21	2.47	2.22		
FEF_{25-75}, L/s	2.72	2.90	2.88	2.67	2.88	2.74	2.82	2.74		
$\dot{V}max$, L/s	5.65	6.10	6.01	5.26	5.46	5.31	5.88	5.32		
$\dot{V}_{25}$, L/s	4.45	4.92	4.80	4.38	4.55	4.34	4.68	4.39		
$\dot{V}_{50}$, L/s	3.12	3.28	3.34	3.15	3.43	3.21	3.23	3.23		
$\dot{V}_{75}$, L/s	1.54	1.67	1.62	1.50	1.66	1.59	1.60	1.56		
Mean residual lung function†										
FVC, L	−0.101	−0.043	0.000	0.032	0.049	0.000			0.12	0.61
FEV_1, L	−0.084	0.004	0.000	0.007	0.065	0.000			0.08	0.29
FEF_{25-75}, L/s	−0.143	0.025	0.000	−0.097	0.118	0.000			0.10	0.05
$\dot{V}max$, L/s	−0.369	0.066	0.000	−0.029	0.167	0.000			0.004	0.37
$\dot{V}_{25}$, L/s	−0.362	0.104	0.000	0.036	0.225	0.000			0.003	0.19
$\dot{V}_{50}$, L/s	−0.213	−0.066	0.000	−0.067	0.212	0.000			0.06	0.03
$\dot{V}_{75}$, L/s	−0.076	0.046	0.000	−0.103	0.057	0.000			0.17	0.05

Definition of abbreviations: FVC = forced vital capacity; FEV_1 = forced expiratory volume in one second; FEF_{25-75} = forced expiratory flow during the middle half of the FVC; $\dot{V}max$ = maximal flow; $\dot{V}_{25, 50}$, and $_{75}$ = maximal flow after exhalation of 25, 50, or 75% of FVC.

* From analysis of covariance, using age, height, and area as covariates.

† From analysis of variance on residuals, calculated from linear regressions obtained in children from nonsmoking households, using age, height, and area as the independent variables.

of age, highly significant differences ($p < 0.004$) were found for indexes of large airways function ($\dot{V}max$ and $\dot{V}_{25}$); a difference of borderline significance ($p = 0.061$) was noted for $\dot{V}_{50}$. For female children, significant differences ($p < 0.05$) were found only for indexes of small airways function ($\dot{V}_{50}$ and $\dot{V}_{75}$), the difference for FEF_{25-75} approaching significance ($p = 0.052$). Results similar to those reported in table 2 from the ANOVA on the residuals were obtained from the covariance analyses.

Linear contrasts were performed on all lung function tests for which the analysis of variance yielded significant differences. The lung function of children in the "only father smokes" category, although higher on the average than that of children in the nonsmoking households, was not significantly different from the latter. Comparing the households where at least the mother smoked to the combined average of those where only the father smoked or no one smoked, significant differences did exist. For male children, the levels of significance were $p < 0.001$ for $\dot{V}max$ and $\dot{V}_{25}$. Female children from households where the mother smoked had a significantly lower residual for $\dot{V}_{75}$ ($p < 0.02$) and a lower residual of borderline significance for $\dot{V}_{50}$ ($p = 0.035$) than children in the other household categories. These p values should be compared to an α level of 0.05/2, or 0.025, because 2 contrasts were made for each analysis (20).

To assess the magnitude of the difference in lung function for children from households where the mother smoked, the percent reduction in mean values was calculated (table 3). The largest reduction for male children is seen in $\dot{V}max$ (8.3%) and for female children in $\dot{V}_{75}$ (6.6%).

In tables 4 and 5, results similar to those presented in table 2 are reported separately for children 7 to 11 for children and 12 to 17 yr of age. There were no significant differences between household types in the slopes of the pulmonary function variables regressed against height and age for the different age-sex groups. For male children 7 to 11 yr of age (table 4), significant differences ($p < 0.03$) were again found for $\dot{V}max$ and $\dot{V}_{25}$, as well as for $\dot{V}_{50}$ ($p < 0.04$). Among the older male children, however, no significant differences across household categories were noted for any lung function test, although the results for $\dot{V}max$ and $\dot{V}_{25}$ approached significance ($p < 0.1$). Note that the sample sizes for the separate age groups are smaller, so that it takes a larger mean difference to obtain significance. The results from the linear contrasts failed to demonstrate any significant differences between the nonsmoking households and those where only the father smoked for any test of lung function. Contrasting the lung function results for households where the mother smoked versus the other 2 categories of households for male children 7 to 11 yr of age, significant differences were found for $\dot{V}max$ ($p < 0.02$) and $\dot{V}_{25}$ ($p < 0.005$).

For the female children (table 5), no significant differences were noted among those 7 to 11 yr of age, whereas FEF_{25-75}, $\dot{V}_{50}$, and $\dot{V}_{75}$ were significant ($p < 0.032$) for those 12 to 17 yr of age. The contrasts for $\dot{V}_{75}$, $\dot{V}_{50}$, and FEF_{25-75} for the older females indicated no significant differences between the means when nonsmoking households were compared with those where only the father smoked. In comparing the households where the mother smoked with the average of the other two, significant differences were found for $\dot{V}_{75}$ ($p < 0.005$) and FEF_{25-75} ($p < 0.02$), and the difference for $\dot{V}_{50}$ approached significance ($p < 0.05$).

To evaluate the possibility that quantity of parental cigarette smoking may be an important determinant of the relationship between passive exposure to tobacco smoke in the household and children's lung function, we regressed lung function residuals of children against the number of cigarettes currently smoked by their mothers (in "at least mother smokes" households) or their fathers (in "only father smokes" households). The results of these analyses failed to reveal any significant correlation between children's lung function and the quantity of tobacco smoked by their parents among either male or female children in either parent-smoking household category.

To assess the effects of passive smok-

TABLE 3

PERCENT REDUCTION IN MEAN LUNG FUNCTION AMONG CHILDREN IN FAMILIES WHERE AT LEAST MOTHER SMOKES

Lung Function Tests	Males	Females
FVC	−3.5*	1.3
FEV_1	−3.4	0.3
FEF_{25-75}	−5.1	−3.5
$\dot{V}max$	−8.3	−0.5
$\dot{V}_{25}$	−7.7	0.8
$\dot{V}_{50}$	−6.6	−2.1
$\dot{V}_{75}$	−4.8	−6.6

For definition of abbreviations, see table 2.

* Represents ratio of mean residual lung function value to adjusted overall mean value, expressed as percent.

TABLE 4

ADJUSTED MEAN LUNG FUNCTION VALUES,* MEAN RESIDUAL LUNG FUNCTION VALUES,† AND SIGNIFICANCE LEVELS† FOR MALE CHILDREN, BY HOUSEHOLD TYPE AND AGE GROUP

	Ages 7 to 11 Yr				Ages 12 to 17 Yr			
Lung Function Test	At Least Mother Smokes	Only Father Smokes	Everyone Non-smoker	Significance Level	At Least Mother Smokes	Only Father Smokes	Everyone Non-smoker	Significance Level
Adjusted mean levels								
FVC	1.95	2.01	2.05		3.65	3.73	3.77	
FEV_1	1.66	1.72	1.72		3.08	3.20	3.20	
FEF_{25-75}	1.94	2.05	2.03		3.43	3.65	3.62	
$\dot{V}max$	3.96	4.17	4.22		7.20	7.74	7.57	
$\dot{V}_{25}$	3.17	3.45	3.44		5.64	6.16	5.98	
$\dot{V}_{50}$	2.26	2.34	2.47		3.88	4.10	4.10	
$\dot{V}_{75}$	1.13	1.13	1.14		1.91	2.13	2.05	
Mean residual levels								
FVC	−0.070	0.031	0.044	0.10	−0.130	−0.102	−0.040	0.51
FEV_1	−0.043	0.060	0.036	0.09	−0.123	−0.041	−0.033	0.37
FEF_{25-75}	−0.083	0.085	0.051	0.18	−0.199	−0.022	−0.045	0.37
$\dot{V}max$	−0.280	0.086	0.112	0.03	−0.451	0.050	−0.099	0.08
$\dot{V}_{25}$	−0.347	0.076	0.070	0.01	−0.376	0.126	−0.062	0.10
$\dot{V}_{50}$	−0.198	−0.060	0.054	0.04	−0.227	−0.070	−0.047	0.45
$\dot{V}_{75}$	−0.000	0.059	0.031	0.74	−0.147	0.036	−0.026	0.21
Number of children	100	44	90‡		107	55	100	

For definition of abbreviations, see table 2.

* From analysis of covariance, using age, height, and area as covariates.

† From analysis of variance on residuals, calculated from linear regressions obtained in children from nonsmoking households, using age, height, and area as the independent variables.

‡ Eighty-eight for FEF_{25-75}, $\dot{V}max$, $\dot{V}_{25}$, $\dot{V}_{50}$, $\dot{V}_{75}$.

ing on children with asthma or bronchitis (n = 138), who were excluded from the main analysis described above, a separate analysis of variance similar to that reported in table 3 was performed on their residual values. No significant differences were found among the male asthmatic and bronchitic children (n = 78) across household categories. Among the female children with asthma or bronchitis (n = 60), significant differences ($p < 0.02$) were found for FEF_{25-75} and $\dot{V}_{50}$; for both of these tests of lung function, however, the children from nonsmoking households had the lowest residuals, and those from households where only the father smoked had the highest. Prevalence of asthma among children did not differ across the different household categories.

For nonasthmatic, as well as asthmatic, children, no significant differences were found in the number of either males or females reporting any *particular* symptom (or *any* symptom) by household type (chi-square test for independence or Fisher's exact test).

Discussion

The present study indicates that passive exposure to at least maternal smoking is associated with reduced lung function in nonsmoking children, but is not related to the occurrence of chronic respiratory symptoms in children. Paternal smoking alone, on the other hand, was not associated with any decrement in children's lung function or with prevalence of respiratory symptoms. One possible, but unproved, explanation for these findings is that children may be more intensively exposed to smoking by the mother, who is more likely to spend more time in the home, especially during the early life of her children, and thus to contribute more to indoor smoke pollution. To further explore this possibility, the work status of the mother was considered. A separate analysis of variance was performed wherein households in which only the mother smokes but works outside the home were contrasted with those in which only the mother smokes and does not work. These contrasts failed to reveal any significant differences in the residual lung function of either male or female children in either the younger or older age category. These findings, however, do not preclude the possibility that a mother's smoking—regardless of work status—has a greater impact on her children than paternal smoking, because even working mothers generally spend more time in the household involved in direct child care activities than do fathers over the entire lifetime of their children.

Our observation of a significant relationship between maternal smoking and decrements in indexes of small airways function among girls 12 to 17 yr of age but not among those 7 to 11 yr of age is consistent with the possibility of either a selective adverse effect of passive smoking on older girls or, more likely, an "active" smoking effect (21) among older girls in the maternal smoking household category who might selectively be underreporting personal cigarette smoking. The latter possibility is suggested by our finding of an unexpectedly low prevalence of self-reported active smoking among teenage girls (12%), including those from smoking households.

In contrast, we observed a more consistent relationship between maternal smoking and decrements in lung function among male children both 7 to 11 yr of age and 12 to 17 yr of age; this relationship was significant for the younger boys and approached significance for the older male children. These findings suggest a passive smoking effect in boys exposed to maternal smoking. The fact that the decrements in lung function in the older male children in the maternal smoking household category did not achieve statistical significance could be due to greater variability in height- and age-adjusted lung function among older than among younger boys. This is consistent with our earlier finding that teenage boys are in the phase of lung growth where the most rapid rates of change in lung function are occurring (22). Moreover, "true" passive smoking effects in older male children might have been confounded and obscured if active smoking behavior were underreported by teenage boys to a comparable extent in all household categories. Finally, older male children from "at least mother smokes" households may be subjected to less passive exposure to maternal smoking than are younger boys because of differences in the proportion of time spent in the house after school.

On the other hand, the discrepancy in the apparent effect of passive smoking on younger male versus younger female children is unlikely to be due to differences in involuntary exposure to second-hand smoke because, if anything, boys are less likely than girls to be exposed to maternal smoking in the household because of differences in indoor-outdoor play activities. This

TABLE 5

ADJUSTED MEAN LUNG FUNCTION VALUES,* MEAN RESIDUAL LUNG FUNCTION VALUES,† AND SIGNIFICANCE LEVELS† FOR FEMALE CHILDREN, BY HOUSEHOLD TYPE AND AGE GROUP

Lung Function Test	Ages 7 to 11 Yr: At Least Mother Smokes	Ages 7 to 11 Yr: Only Father Smokes	Ages 7 to 11 Yr: Everyone Non-smoker	Significance Level	Ages 12 to 17 Yr: At Least Mother Smokes	Ages 12 to 17 Yr: Only Father Smokes	Ages 12 to 17 Yr: Everyone Non-smoker	Significance Level
Adjusted mean levels								
FVC	1.92	1.94	1.86		3.15	3.17	3.16	
FEV_1	1.64	1.65	1.60		2.75	2.85	2.77	
FEF_{25-75}	2.00	2.02	2.00		3.31	3.68	3.43	
$\dot{V}max$	3.92	4.13	3.88		6.55	6.70	6.67	
$\dot{V}_{25}$	3.32	3.47	3.29		5.35	5.58	5.33	
$\dot{V}_{50}$	2.39	2.50	2.37		3.87	4.29	4.02	
$\dot{V}_{75}$	1.15	1.15	1.15		1.84	2.13	1.99	
Mean residual levels								
FVC	0.044	0.056	−0.018	0.47	0.020	0.044	0.019	0.94
FEV_1	0.017	0.023	−0.013	0.74	−0.002	0.099	0.013	0.25
FEF_{25-75}	−0.090	−0.075	−0.029	0.80	−0.103	0.274	0.030	0.02
$\dot{V}max$	−0.031	0.153	−0.050	0.50	−0.026	0.178	0.051	0.64
$\dot{V}_{25}$	−0.002	0.104	−0.047	0.86	0.071	0.322	0.049	0.31
$\dot{V}_{50}$	−0.100	−0.002	−0.060	0.75	−0.035	0.384	0.064	0.03
$\dot{V}_{75}$	−0.032	−0.037	−0.014	0.95	−0.169	0.132	0.014	0.01
Number of children	97	42‡	93‡		103‡	51	89‡	

For definition of abbreviations, see table 2.

* From analysis of covariance, using age, height, and area as covariates.

† From analysis of variance on residuals, calculated from linear regressions obtained in children from nonsmoking households, using age, height, and area as the independent variables.

‡ 41, 92, 102, and 88, respectively, for FEF_{25-75}, $\dot{V}max$, $\dot{V}_{25}$, $\dot{V}_{50}$, $\dot{V}_{75}$.

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sex-related discrepancy in the younger children could possibly be due to inherently greater vulnerability of boys than girls to the injurious effects of inhaled respiratory irritants. The prevalence of asthma is greater in males from birth up to 16 yr of age (23). It is possible, therefore, that more boys than girls in our study sample had latent asthma. Such boys may have had relatively greater airways reactivity to the smoke contaminating their indoor environment, leading to a sustained deficit in lung function.

Our failure to observe a significant relationship between the current quantity of smoking by parents and the lung function residuals of their children suggests that other factors relating to parental smoking may have a more important influence on children's lung function. Such factors (not evaluated by us) could include variations in room ventilation, in previous levels of cigarette smoking by parents (when their children were younger), and in the smoking habits of parents in the presence of their children.

Factors that need to be considered as possible confounders of the relationship between parental smoking and children's respiratory status include variations across household categories in (*1*) the type and level of community air pollution, (*2*) non-cigarette-related indoor pollution (such as that caused by gas cooking) (24–26), (*3*) crowding, and (*4*) socioeconomic status. The households examined in this report were located in 4 geographically distinct areas of Southern California, which were selected because of their similarity in demographic distributions, including race, income, and home value, and their historical dissimilarity in type and/or level of atmospheric pollution: low levels of chemical pollutants (Lancaster), moderate to heavy levels of oxidant pollution (Burbank and Glendora, respectively), and moderately heavy levels of SO_2, hydrocarbons, and particulates (Long Beach). Cross-sectional comparisons of lung function of adults across these communities have shown significant differences in age-, height-, and sex-adjusted mean values for different indexes of lung function, the worst mean values being noted in the residents of the communities with the worst air pollution (Glendora and Long Beach) (16, 17).

In analyzing the relationship between parental smoking and children's lung function, we believe that possible effects of ambient air pollution on children's lung function were adequately controlled for through adjustment for the effects of area by using area as a dummy variable in our linear regression equations and as a covariate in our analyses of covariance. Moreover, the proportion of parent-smoking households was not significantly different across all 4 study areas and, if anything, was actually highest in our community with the cleanest air.

Although cigarette smoke probably constitutes the most important source of indoor air contamination, other indoor pollutants, particularly gas fuel used in cooking, might adversely affect lung function; studies of the impact of indoor exposure to cooking fuel on lung function, however, have yielded variable results (26, 27). Questions concerning the mode of indoor cooking were not asked of our subjects until the second round of testing in our cohort study. Data collected subsequently from 200 of the 615 households (33%) in our cohort indicate that during the lifetime of children included in the analysis, 102 households used gas fuel alone for cooking, 20 used electricity alone, and 78 had switched from one type of fuel to the other at least once. The proportion of households in which gas fuel alone was used for cooking was similar across parental smoking household categories. Moreover, using these data in a two-way analysis of covariance, we failed to find a significant effect of exposure to gas cooking fuel on lung function or any interactive effect of gas cooking and passive smoking on lung function.

Although the Hollingshead score for occupation of the head of household (an indicator of socioeconomic status) was related to household smoking status category, it showed no relationship to children's lung function, nor was there a significant interaction between the effect of household category and the Hollingshead score on children's lung function (two-way ANOVA). Parental level of education (another socioeconomic indicator), although significantly higher in nonsmoking households, did not correlate with children's residual lung function. The size of the families and the number of rooms in the residence were evenly distributed across household categories, decreasing the possibility that our results were confounded by any effects of crowding or of volume of dilution of indoor pollutants on lung function. Although the exclusion of some children because of missing or technically unsatisfactory data could have biased our results, the observation that the proportion of such children was evenly distributed across household categories and that the excluded children were of a similar age and height to those of the children included in the analysis should have reduced these biases.

Even though the risk of chronic pulmonary alterations caused by passive tobacco smoke exposure may be relatively subtle and only variably demonstrable in the general population (10, 12, 27, 28), passive smoking might be expected to have more obvious respiratory consequences in asthmatics, who hyperreact to a number of noxious stimuli. In all 4 of our study areas, the prevalence of asthma or bronchitis was similar among children in the different household categories: at least mother smokes (11%), father only smokes (13%), and no parent smokes (10%). However, no relationship was apparent between parental smoking and either lung function or symptomatology of asthmatic boys; moreover, among female children with asthma, the *lowest* residuals were actually noted in those from households in which neither parent smoked, and this relationship was significant ($p < 0.02$). Although these unexpected findings do not support the hypothesis that parental smoking aggravates children's asthma (29), the exclusion of households with ex-smoking parents from our analysis could have biased our results.

Our finding of lower lung function in nonsmoking boys from households with at least maternal smoking is somewhat in accord with, and extends, the data of Tager and coworkers (12), who found a relationship between the smoking habits of parents and the midexpiratory flow rates of their children 5 to 9 yr of age. The latter investigators, however, did not examine the impact of parental smoking on male and female children separately, so that the trend they noted toward a decreasing level of function in children with increasing parental smoking might have been stronger or weaker if the results in boys and girls had been analyzed separately. Also, it would have been of interest if they had analyzed other indexes of lung function in addition to FEF_{25-75} because the strongest associations we noted were between parental smoking and the residuals for Vmax and V_{25} in

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boys. Moreover, we excluded ex-smokers from analysis, whereas their definitions of parent-smoking households accepted parent ex-smokers as "current" smokers if they had smoked during the first year of life of all their children. Therefore, if passive smoking effects on children are influenced by persistent exposure to smoke contamination in the household beyond the first year of life, the effect of parental smoking noted by these investigators might have been underestimated.

On the other hand, our results are not in agreement with those of Dodge (27) or of Schilling and associates (10). Dodge was unable to demonstrate a significant relationship between the smoking of either Anglo-white or Mexican-American parents and the FEV_1 of their Arizona schoolchildren (27). In our study, we were also unable to find a significant relationship between the FEV_1 of children of either sex and the smoking of their parents, but this relationship approached significance in boys ($p < 0.1$). The discrepancy between our findings and those of Schilling and associates (10), who failed to find a significant contribution of parents' smoking to children's lung function in 3 towns in Connecticut, may be due to differences in sample sizes, types of lung function examined, or analytic techniques. Also, it is not clear how these investigators classify parent ex-smokers, who were excluded from our analysis. Classification of ex-smokers as either smokers or nonsmokers could conceivably obscure significant differences between parent-smoking and lifetime nonsmoking households.

Although we found significantly lower values for Vmax and V_{25} among boys in relation to maternal smoking, these decrements were, in fact, relatively small (e.g., 7.7%, or 0.37 L/s, decrease in V_{25}). Although these decrements were statistically significant, their biologic significance might be questioned. In this regard, it is noteworthy that the children we studied, especially the younger children, were undergoing a rapid rate of growth and development of their lungs. Consequently, the small difference in lung function we observed could be important if it interfered with the normal achievement of full lung growth, as previously suggested (26).

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